SECTION III:
Life and Death in Colonial New York

Chapter 8

Childhood Health and Dental Development

M.L. Blakey, M. Mack, A.R. Barrett, S.S. Mahoney and A.H. Goodman

Dental enamel hypoplasias are defects in crown development that appear as a transverse groove or series of pits that are partially or entirely around the circumference of the tooth. Hypoplastic defects, while they manifest in the teeth, result from metabolic disturbances of malnutrition and disease elsewhere in the body. Enamel hypoplasias thus provide evidence of general stress that may have been brought about by many different kinds of stressors. Like other "general stress indicators" such as life expectancy, infant mortality, or growth retardation rates, frequencies of hypoplastic defects can be compared among different populations as a gross index of physical well-being and the adequacy of societal resources upon which the physical quality of life may depend. Of particular value, enamel hypoplasia develops in childhood and adolescence when both the deciduous and permanent teeth are formed.

The evidence of these early stresses remains apparent in adult skeletons in which teeth have been retained. The defects occurring on different teeth and in different locations on teeth represent stresses at differing ages during childhood and adolescent growth, similar to the analysis of tree rings for a record of droughts during the lifetime of a tree. These defects have been observed in archaeological collections and living populations representing a very broad range of human experiences, from those of early hominids to industrial nations. Included among these are a number of studies from

African-American and Afro-Caribbean archaeological sites (Blakey and Armelagos 1985; Blakey et al. 1994; Goodman et al. 1984; Goodman and Armelagos 1985; Condon and Rose 1992; Corruccini et al. 1985; Clarke 1982).

This chapter puts forward an analysis of hypoplasia frequencies in the African Burial Ground (ABG) sample. Comparisons are made of enamel defect frequencies in different age groups and sex/gender groups. We compare individuals with culturally-modified teeth who were probably born in Africa and those with unmodified teeth whose origins are unknown. We also compare the New York sample with skeletal collections from other diasporic archaeological sites. Questions regarding the physical quality of life in childhood are central as is our assessment of these data for evidence of health differences or transitions among Africa, the Caribbean, and New York, which take place at different points in the life cycles of New York Africans.

Deciduous dental enamel begins to develop during the fifth month in-utero, completing development by the end of the first year of postnatal life. Permanent dental enamel begins formation at birth and continues into the sixteenth year of age. General stress indicators are visible in dental enamel due to the process of enamel formation. Ameloblastic (enamel producing) activity involves cellular production of a protein rich matrix that mineralizes, forming the crystalline enamel of teeth. If the development of the enamel crown is interrupted by physiological insult, a transverse groove or series of pits (hypoplasia) or discolored enamel (hypocalcification) results in the 'rings' of enamel being laid down at that time (see Figure 8.1 and Figure 8.2).

Hypoplasia results from differential thickness in the enamel, whereas hypocalcification occurs during interruption within the final stages of ameloblastic

activity, and results in discoloration of the tooth enamel (Blakey et al., 1994, p. 372). Dental enamel is acellular and, therefore, lesions and discolorations due to physiological stress are permanent and are not obliterated through cellular renewal. In

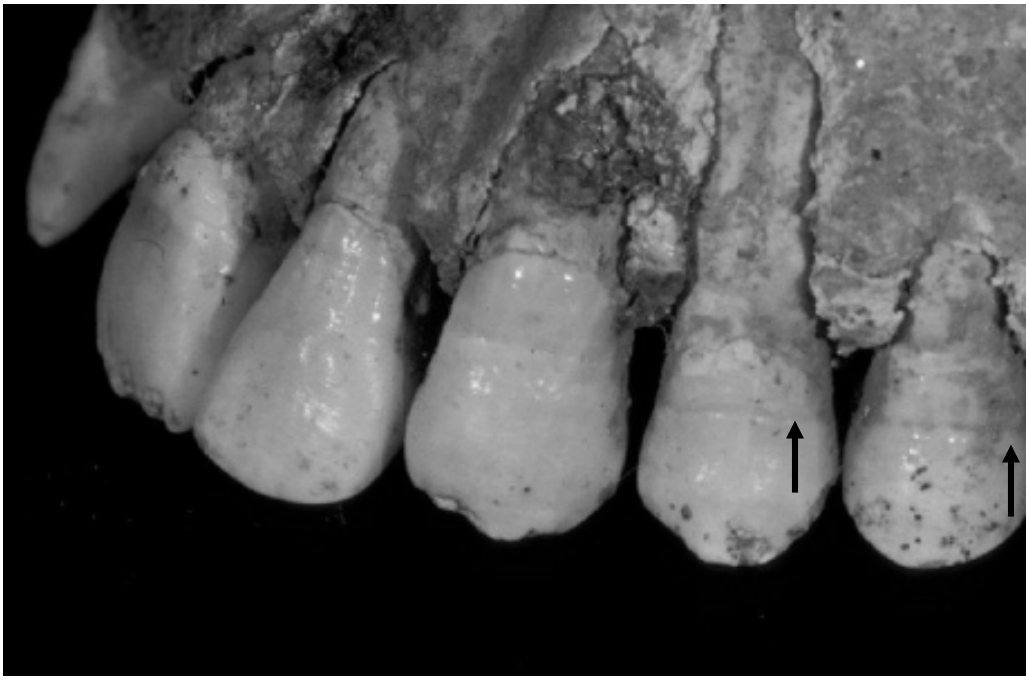

Figure 8.1: Linear enamel hypoplastic lesions in the anterior maxillary permanent dentition in a female aged 20 – 25 years (Burial 1)

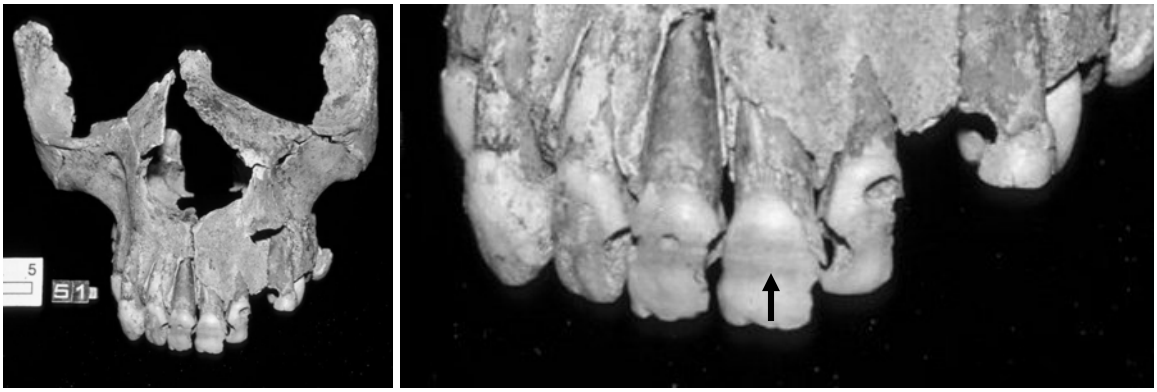

Figure 8.2: Bands of discoloration caused by hypocalcification in the anterior maxillary permanent dentition in a 24 – 32 year old female (left), magnification (right) (Burial 51)

addition to general identification of stress incidence during enamel formation, the rate of enamel matrix formation provides a mechanism for estimating the developmental stage at which the growth arrest occurred (Blakey et al. 1994:372; Goodman and Armelagos 1985). Hypoplasia provides an estimation of stress severity and/or duration by the size of the malformation. With rare exception, dental enamel hypoplasia is a result of systemic metabolic stress associated with infectious disease, insufficient calcium, protein, or carbohydrates, and low birth weight, characterized together as "general stress" (Goodman et al. 1988; Blakey et al. 1994).

Materials and Method

A subsample was selected from the ABG sample to study the occurrence and frequency of hypoplasia within adults and children (see Table 8.1). Within this study, the presence of hypoplasia within an individual was defined by the presence of linear or non-linear hypoplasia in one of the teeth selected for analysis. The absence of hypoplasia was

Table 8.1: Summary of Study Samples

Study description	Dentition	Samples Size	
Hypoplasia and Hypocalcification	Canines and Incisors - Permanent	65	99
Hypoplasia and Hypocalcification	Canines and Incisors - Deciduous	34	
Hypoplasia Controlled for Attrition	Canines and Incisors - Permanent	48	
Hypoplasia Controlled for Attrition	Third Molars	97	
Canine Chronology for Hypoplasia	Canines - Permanent	23	
Hypoplasia and Hypocalcification	Third Molars	111	

defined by the absence of hypoplasia in all teeth selected for analysis. According to research conducted by Goodman and coworkers, secondary canines and incisors display

95 percent of enamel hypoplasia observed where all available dentition is represented (Goodman et al. 1980). The current study employed this "best tooth" method in selecting individuals with the presence of a permanent left or right maxillary central incisor and a left or right mandibular canine. The presence for permanent teeth was defined according to codes 2 and 7 within *Standards* (Buikstra and Ubelaker 1994) indicating that teeth are fully developed, in occlusion and observable. A total of 65 individuals within the ABG were selected for analysis of permanent dentition which represents the developmental period between birth and 6.5 years of age. A separate selection was conducted for individuals with permanent third molars, left or right, mandibular or maxillary, where presence was defined by codes 2 and 7 within *Standards* (Buikstra and Ubelaker 1994: 49). One hundred and eleven individuals are included within this third molar analysis, which represents the developmental period in life from nine years to approximately sixteen years of age.

A subsample was selected from the permanent canine and incisor study and from the third molar study to control for age- or sex-related differences in dental attrition that might affect hypoplasia frequencies. Individuals with moderate to severe dental wear and individuals for whom dental wear could not be scored (including inability to score due to cultural modifications such as filing and pipe notches), were removed from the canine and incisor sample and from the third molar sample. Individuals with a dental wear score of five or greater, according to Smith (1984), were removed from the permanent incisor and canine sample, resulting in 48 observable individuals. Individuals with a dental wear score of seven or greater, according to Scott (1979), were removed from the third molar sample, resulting in 97 observable individuals.

Deciduous dentition was studied by selecting individuals older than one year with one left or right central maxillary incisor, one left or right mandibular canine, and one second molar (see Figure 8.3). The presence for deciduous teeth was defined by codes 1, 2 and 7 within the *Standards* where the teeth were fully developed and observable. Thirty-four subadults were selected to assess hypoplasia in deciduous dentition. Developmental stages spanning approximately five months in-utero to sixteen or seventeen years of life are represented by the dentition selected for analysis within this study. Statistical analysis for each study employed SPSS software version 11.5.

Figure 8.3: Deciduous mandibular dentition with a single non-linear hypoplastic pit in the right canine of a subadult aged 3 – 5 years. This individual also appears to have been anemic (Burial 7)

Twenty-three individuals were assessed for the chronology of physiological stress episodes resulting in hypoplastic lesions. Chronology was determined for defects in the

left permanent mandibular canines; however, right mandibular canines were used when the left was absent or unobservable. Measurements for the hypoplastic lesion's beginning and ending had been recorded by members of the ABGP in the late 1990s (see Figure 8.4). The distance from the dental cervix to the onset of the incisal (beginning) aspect of the lesion was recorded, followed by the measurement of the cervical (latest developing) aspect of the lesion. A midpoint for this episode was calculated, and this measurement was used in conjunction with the total crown height measurement to estimate the age at which each episode occurred.

Figure 8.4: Permanent mandibular canine and lateral incisor with linear Hypoplasia in a male aged 35 – 45 years (Burial 9)

Total crown height was divided by the number of years the mandibular canine develops (6 years), and this figure served as an index representing an increment of growth in one year. The midpoint measurement was divided by the yearly incremental

growth index, which provided the number of years prior to the end of enamel development (6.5 years of age) at which the incident occurred. Next, this figure was subtracted from 6.5 to arrive at the age of occurrence for each episode. For analysis within this study, the midpoint of the canine, representing the developmental period of 3.5 years, was calculated for each tooth. Episodes were coded as occurring before 3.5 years and after 3.5 years (see Table 8.2). Three and a half years is also the age at which central incisal crown development ends, providing a comparison of frequencies represented between the incisor and canine and between the correspondent ages of crown development within the canine.

Table 8.2: NYABG Canine Chronology Formula and Example Calculation: CH/6 = YGI 6.5 – (MID/YGI) = Age of Occurrence

Crown Height (CH) (mm)	Total Years of Development	Yearly Growth Increment (YGI)	Crown Midpoint at 3.5 years	Hypoplastic Lesion Midpoint (mm)	Formula	Age of Occurrence (years)
12.71	6	12.71/6=2.12	6.36	3.93	3.93/2.12=1.85	6.5 – 1.85=4.65

Results

Among the 65 individuals with permanent dentition, 70.8 percent were hypoplastic. Frequencies for hypoplasia in permanent dentition are higher in the ABG sample than those observed in the enslaved populations of Catoctin Furnace, Maryland (Kelley and Angel 1987) or Newton Plantation in Barbados (Corruccini et al. 1985). The New York frequencies are lower than the total frequencies observed in the largely free and freed nineteenth-century Philadelphia First African Baptist Church (FABC) sample (Blakey et al. 1994) or enslaved African Americans buried in nineteenth-century Charleston, South Carolina, 38CH778 (Rathbun 1987). The difference in hypoplasia frequencies may reflect the time trajectories and geographic locations represented within

these populations. A greater number of people within the ABG and Barbados sites more likely would have been born in Africa than would have been the case for the nineteenth-century African-Americans in Philadelphia and the South. The latter group spent their lives within the conditions of slavery or as free people living under conditions of economic and social inequality.

The difference in hypoplasia frequencies for men and women in the ABG was not statistically significant [62.5% of the women (n=15) and 74.3% of the men (n= 26)], indicating that male and female children experienced similar frequencies of stress episodes from birth to the age of 6.5 years. However, the New York ABG sample does fall into the general pattern established by previous studies (mentioned earlier and here) indicating that the men have consistently higher percentages of hypoplasia than females (Rathbun 1987; Owsley et al. 1987; Khudabux 1991). Blakey and coworkers (1994) report 86 percent hypoplasia in women and 92 percent in men among 54 individuals from the FABC sample. Angel and coworkers report 71 percent of men and 43 percent of the women at Catoctin Furnace, Maryland, had hypoplasia. The Blakey et al. (1994) study of the Catoctin site indicates that women had higher frequencies of slight linear enamel hypoplasias; however, men had a greater frequency of moderate to severe hypoplasias [68 percent males (n=17) and 37.9 percent females (n=11)]. Among the populations compared within this study, Rathbun (1987) reports the highest frequencies in men and women at the Charleston, South Carolina site (71 percent in women and 100 percent for men). Tables 8.3 and 8.4 provide comparative frequencies and other data for the studies just discussed, while frequency data for the NYABG sample are presented in Table 8.5.

Table 8.3: Frequency of Hypoplasia in Males and Females at NYABG N=59*

Males n=35		Females n=24	
Present	**Absent**	**Present**	**Absent**
74.3% (n=26)	25.7% (n=9)	62.5% (n=15)	37.5% (n=9)

*6 of the 65 individuals with adult dentition were too young to determine sex. Therefore, these individuals are not represented in the total number of males and females.

Table 8.4: Comparison of Frequencies Reported in Skeletal Populations
***frequencies were not reported for this category**

Site/ Location	Region	Rural/ Urban	Historical Period	Hypoplasia Frequency/ secondary dentition (%)	Hypoplasia in Females (%)	Hypoplasia in Males (%)	Hypoplasia in Subadults/ deciduous dentition (%)
NYABG, New York	Northeast, North America	Urban	17th and 18th centuries	70.8 (n=46)	62.5 (n=15)	74.3 (n=26)	85.3 (n=34)
Newton Plantation, Barbados	Barbados, West Indies	Rural	1650s - 1834	54.5 (n=56)	*	*	*
FABC, Pennsylvania	Northeast, North America	Urban	1800 - 1850	89 (n=54)	86 (n=29)	92 (n=25)	92.5 (n=30)
Catoctin Furnace, Maryland	Southeast, North America	Urban	1790 - 1820	46 (n=7)	43 **Slight** 79.3* (n=23) **Mod-Sev** 37.9* (n=11)	71 **Slight** 68* (n=17) **Mod-Sev** 68 (n=17)	*
Charleston, S. Carolina (38CH778)	Southeast, North America	Rural	1840 - 1870	85 (n=27)	71 (n=14)	100 (n=13)	*

Sources: Newton Plantation site frequencies from Corruccini et al. (1985), First African Baptist Church frequencies reported from Blakey et al. (1994) with frequencies in children cited from Rankin-Hill (1997). Catoctin site frequencies reported from Kelley and Angel (1987) for overall frequencies. Frequencies by sex for Catoctin Furnace are from Angel et al. (1987) and Blakey et al. (1994). Frequencies reported by Blakey et al. (1994) have an asterisk (*) and represent frequencies of slight hypoplasia or moderate to severe hypoplasia within the Catoctin Furnace site. Frequencies for males and females in the South Carolina 38CH778 site from Rathbun (1987). Combined secondary dentition frequency calculated from male and female frequencies reported by Rathbun.

Table 8.5: NYABG Frequency of Hypoplasia by Age Group (N=99)

Age Group	Within Age Group		
		Men (n=35)	**Women (n=24)**
1 – 14* (n=37)	86.5% (n=32)		
15 – 24 (n=17)	76.5% (n=13)	83.3% (n=5)	75.0% (n=6)
25 – 55 + (n=45)	66.7% (n=30)	72.4% (n=21)	56.3% (n=9)

*Three children within this age category had permanent dentition

Among the 99 ABG individuals within the canine and incisor study, 37.4 percent (n=37) died before the age of 15 years, 86.5 percent (n=32) of whom had hypoplasia. Young adults who died between the ages of 15 and 24 years of age represent 17.2 percent of the population, 76.5 percent of whom had hypoplasias. A total of 45.5 percent of the people died after the age of 25 years (n=45), 66.7 percent (n=30) of whom had hypoplasia. The frequency of childhood growth disruption is lowest in the oldest age-at-death groups.

Most of this sample experienced generalized stress in their childhood years. Individuals with permanent dentition (n=65) representing the period of childhood between birth and 6.5 years of age had hypoplasia in 70.8 percent (n=46) of the cases, overall. Notably, this frequency is about 20 percent lower than that for the Philadelphia FABC remains. Among children with deciduous dentition, 85.3 percent of the children (29 of 34) had hypoplasia, representing disrupted development between the fifth month in-utero through the end of the first year of life. In contrast with the permanent dentition findings, this frequency is more than 20 percent higher than for the FABC.

If the FABC can serve as an operational reference point, one can ask why it is that the childhoods of those who died as adults in New York are relatively less stressed, while those who died as children in New York are relatively more stressed, in comparison with the Philadelphians who died in the 1830s and 1840s. The interpretation of this issue bears on the specific histories of in-migration in the two cities that will be addressed later in this chapter.

The foregoing data suggest that the individuals who experienced early stress episodes resulting in enamel hypoplasia were more likely to have died in childhood and that enslaved children in colonial New York experienced high levels of stress. The lower frequency of individuals with hypoplasia among those who were older than age 25 at death may reflect the forced migration of enslaved men and women arriving in colonial New York. These individuals seem more likely to have experienced childhood stress episodes in Africa than in New York, and their lower defect frequencies might reflect childhood experiences elsewhere. The brisk importation, low fertility, and high child mortality of eighteenth-century New York meant that an African who lived there as an adult was more likely to have been born in Africa (or possibly the Caribbean) than to have been born and survived to adulthood in New York. Although some children were imported, those who died as children in New York seem more likely to have been born there than those who died there as adults. Hypoplasia frequencies in the dead children, therefore, seem most likely to reflect the conditions of New York. The data on lead and strontium content in teeth (see Chapter 6) are supportive of those assumptions about the nativity of young children.

Those who died between 15-24 years of age have intermediate frequencies of defects in the teeth that developed during early childhood, as shown in Figure 8.5. We also examined frequencies of hypoplasia in third molars that developed between 9 and approximately 16 years of age. The late childhood and adolescence stress represented by hypoplastic third molars was present in 44.4 percent (n=12) of those who died between 15-24 years and was present in only 10.7 percent (n=9) of those who died at 25 years of age and older in whom we could observe third molars. These differences were

statistically significant (Pearson chi-square with Yates Continuity Correction = 13.035, 1 df, p <.0005). Interestingly, the 15-24 year olds would have died quite close to the time when these late stresses were occurring. The analysis of 111 individuals with third molars was conducted apart from our usual analysis of incisors and canines. The third molars are less sensitive to hypoplasia than are the anterior teeth and cannot be directly compared with them, although the hypoplastic lesions may represent more severe episodes of stress (Goodman and Armelagos 1985).

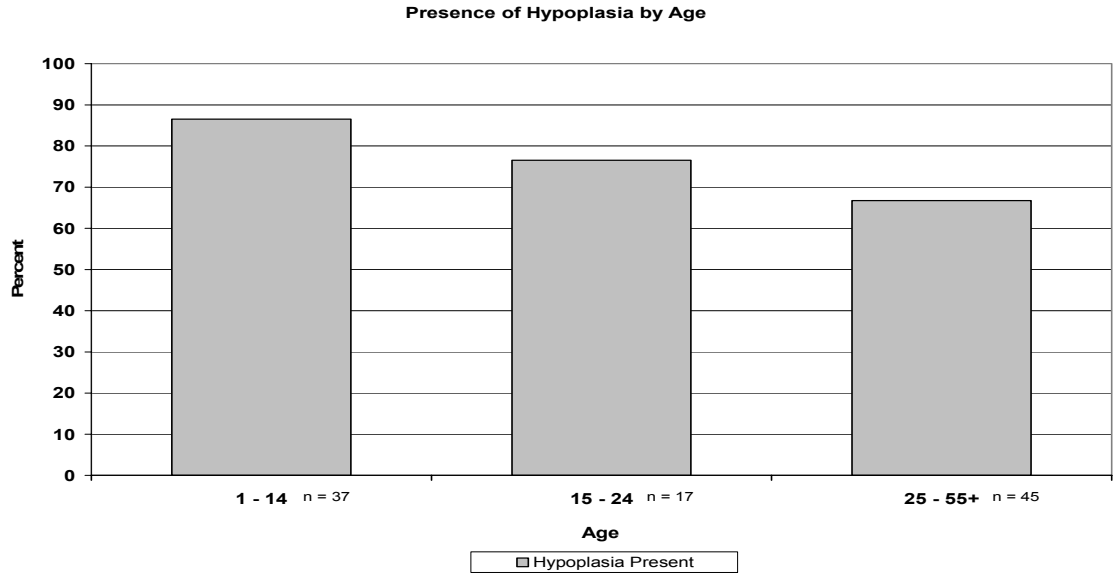

Figure 8.5: NYABG Presence of Hypoplasia by Age (n=99)

A comparison of only third molar data is considered next (see Figure 8.6). We suspect, based on historical documentation of importation ages, that many of the 15-24 year olds are likely, because of age, to have been new arrivals through the trade in human captives, with the Middle Passage constituting another plausible stressor for them. Fifteen years of age was also the beginning of adulthood in most eighteenth-century censuses in New York (10 years of age was the criterion of adulthood less frequently

used). Studies of active periosteal lesions in this group show more new infection in the 15-24 year age range than among the older individuals who exhibit a preponderance of sclerotic and healed lesions. Mortality is also very high among the 15-24 year old males and females, as is detailed in other chapters. Changing conditions of life either through forced migration or/and adult status may be involved in these effects.

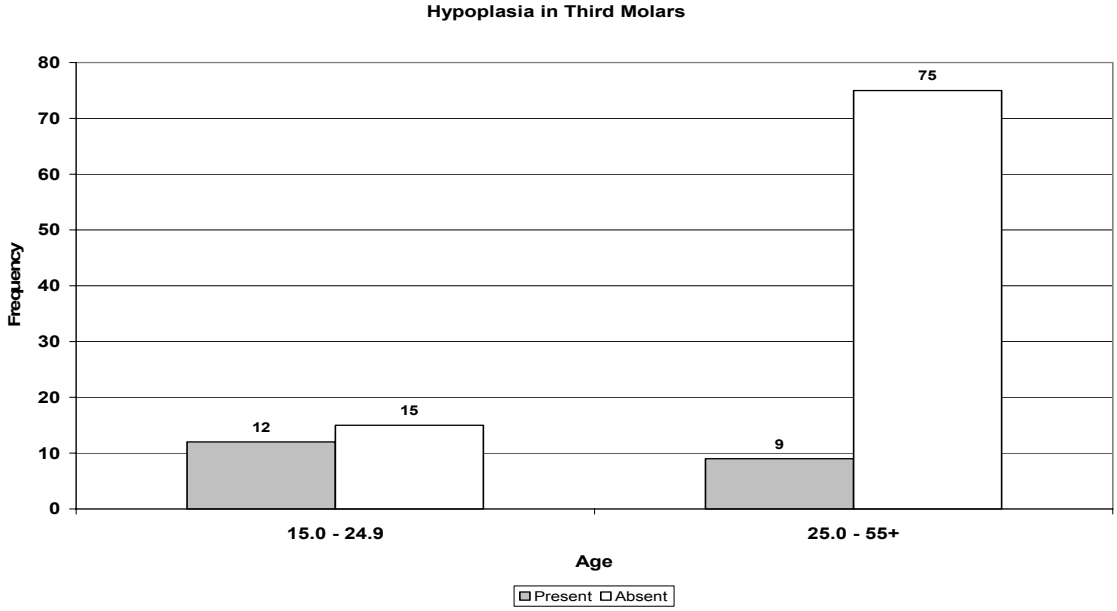

Figure 8.6: NYABG Hypoplasia in Third Molars (n=111)

The skewing of subadult nativity toward New York as the place of birth, and the skewing of adult nativity toward West and Central Africa may help explain low frequencies of hypoplasia in adults and high frequencies in subadults, when compared to nineteenth-century Philadelphians. The FABC, conversely, shows relatively low frequencies in subadults and high frequencies in adults. This may also be related to different places and conditions of childhood for those who died as children and those who died as adults in Philadelphia, since African births probably were not a major factor in mid-nineteenth-century Pennsylvania. Among the FABC sample, subadult nativity

should be skewed toward Philadelphia, as similarly those who died as children in New York were also often born there. Philadelphia in the mid-nineteenth-century can be characterized as having a free, disenfranchised, predominantly impoverished, unskilled wage laboring black community. There was mobility toward greater economic stability among some blacks in the early part of that century, but this was halted during a peak period of Irish immigration into the city at about the time the FABC cemetery was in use (Du Bois 1899; Rankin-Hill 1997). These conditions were stressful, yet hypoplastic stress effects in these dead Philadelphian children were less frequent than in the enslaved children of colonial New York City.

The FABC adults, however, contained a large number of persons who were born and raised in bondage both in late eighteenth-century slave-holding Pennsylvania and on the eighteenth and nineteenth-century Southern plantations from which they were given manumission, bought their freedom, or escaped to Philadelphia (Rankin-Hill 1997).
For these FABC adults, their hypoplastic indicators of childhood stress were higher, relative to those who died as New York Africans but whose childhoods were frequently spent in Africa. This interpretation of the data is assisted by the facts that the same researchers (and methodological training) were involved in both studies, both archaeological samples are sizable, both primary and secondary dentition were observed, and both sites are in of the urban Northeast, thus greatly improving the reliability of comparisons.

Since much of this interpretation relies on the relation of hypoplasia frequency to age, one should examine the extent to which age-related occlusal wear might play a role in reducing our ability to observe hypopoplasias, thus reducing the count of defects in

older individuals. Subsets of the permanent dentition samples were created to control for the possible effect of dental attrition on hypoplasia frequencies between age and sex groups due to loss of observable data through tooth wear. The incisor and canine study, as well as the third molar study, displayed the previously reported pattern of hypoplasia frequencies when attrition was controlled. The highest frequencies were found in individuals aged 15 – 24 years and lower frequencies were found in individuals who lived to be 25 years of age and older. These differences were statistically significant in the third molar analysis only (Pearson chi-square with Yates Continuity Correction = 10.678,1 df, $p < .002$). Men continued to have higher frequencies of hypoplasia than women within both age groups in the canine and incisor study. These gender differences were not statistically significant. Table 8.6 and Table 8.7 provide a summary of hypoplasia frequencies within each study. These findings show that the observed decrease in hypoplasia frequencies for older age groups and the differential frequencies between men and women were not a result of lost data due to tooth attrition.

Table 8.6: NYABG Frequency of Hypoplasia by Age and Sex
Canines and Incisors (controlling for attrition)
(N=48)

Age Group	**Frequency within Age Group**		
		Men (n=24)	**Women (n=21)**
15 – 24 (n=16)*	81.3% (n=13)	100% (n=5)	75.0% (n=6)
25 - 55 + (n=32)	71.9% (n=23)	65.2% (n=15)	34.8% (n=8)

*Three individuals with adult dentition were too young to determine sex. Thus, these individuals are not represented in the total number of males and females.

Table 8.7: NYABG Frequency of Hypoplasia by Age Group
Third Molars (controlling for attrition)
(N=97)

Age Group	Frequency within Age Group
15 – 24 (n=26)	46.2% (n=12)
25 - 55 + (n=71)	12.7% (n=9)

Hypoplasia chronology was calculated for the canines of 23 individuals for whom measurements of hypoplasia lesions were available in the permanent dentition study. Differential frequencies of hypoplasia within permanent canines and incisors were observed, indicating the greater occurrence the experience of life stressors across the age ranges. While 27individuals showed hypoplasia in maxillary central incisors and 40 showed hypoplasia in mandibular canines, variability in sensitivity made direct comparisons questionable.

Canine chronology indicates that of the 23 individuals for whom chronological assessment was possible 87 percent (n=20) experienced stress episodes between 3.5 and 6.5 years of age, versus 21.7 percent (n=5) who experienced episodes in the first 3.5 years of life. Four children (17.4%) exhibited evidence of multiple episodes between birth and 6.5 years of age. The difference in hypoplasia frequencies displayed for the incisors and canines was 21 percent, whereas the difference for hypoplastic occurrence from birth to 3.5 years and 3.5 to 6.5 years is over 50 percent if the incidents occurring in both developmental periods are pooled (see Table 8.8 and Table 8.9). The differential stress pattern between birth to 3.5 years and 3.6 to 6.5 years represented by an individual tooth analysis corresponds to the differential frequencies assessed using chronology within the canine. However, the canine chronology provides a more precise understanding of the ages at which stress episodes occur and of differential stress levels

experienced within the first 3.5 years, versus 3.5 – 6.5 years of age, and is therefore methodologically preferable to the between tooth method.

Table 8.8: NYABG Occurrence of Hypoplasia by Age Range

Age Range	% Hypoplastic
.5 – 3.5 years	21.7% (n=5)
3.6 – 6.5 years	87% (n=20)
0 – 3.5 and 3.6 to 6.5	17.4% (n=4)

Table 8.9: NYABG Occurrence of Hypoplasia by Age Intervals

Age	% Hypoplastic
.5 to 1 year	0
1.01 to 2 years	0
2.01 to 3 years	21.7% (n=5)
3.01 – 4 years	26.1% (n=6)
4.01 – 5 years	87% (n=20)
5.01 – 6.5 years	21.7% (n=5)

Another factor that must be considered in interpreting the chronology is variability of susceptibility within tooth types. The study of the Dickson Mound Population conducted by Goodman and Armelagos (1985) indicates that incisors are more likely to display hypoplasia between the period representing ages 2 to 2.5 years, whereas canines are more likely to display hypoplasia in the fifth to sixth year of development. This suggests that the within-tooth chronological pattern of hypoplasia frequency differences is at least partly a reflection of canine sensitivity patterns.

The individuals within the age category of 1 to 14 years are more likely to have been born in New York than individuals who were older at the time of death. Their early deaths and high levels of stress indicators, such as hypoplasia, support an interpretation that these children were born into the arduous conditions of enslavement and therefore experienced greater levels of diseases and illnesses, possibly a consequence of being

forced to work at young ages. The peak frequencies of hypoplasia between the ages of 3 to 4 years in secondary dentitions observed by Corruccini et al. (1985) were attributed to weaning at ages 2 to 3. Blakey et al. (1994) tested the weaning hypothesis within African-American enslaved groups to argue that enslaved children experience physiological stress from multiple sources, and that weaning does not account for the peak in hypoplasia frequencies. Furthermore, Blakey's study suggests the need for historical and cultural contexts to be considered within a biocultural interpretation. The high frequencies of hypoplasia occurrence during the fifth year demonstrate that this stage was a vulnerable and stressful age for children who survived early infancy and who died as adults. This window on childhood appears to be most pertinent for those who were born in Africa, while childhoods in the Caribbean, New York, and other locations are doubtlessly mixed into our adult sample. How much more stressful the fifth year of age was compared to earlier ages, however, has not been confirmed using enamel defects due to variation in hypoplastic sensitivity across different parts of the crown. Moreover, these data represent the experiences of survivors, while the high death toll of infants clearly represents vulnerability and stress among those who did not survive to exhibit developmental defects in secondary teeth. Those deaths (see Chapter 7) clearly resulted from conditions in New York City, albeit precipitated partly by the poor health of captured mothers whose own experiences of childhood stress were relatively less frequent.

The project has used a political-economic framework for explaining biological variations in the ABG sample. For example, Susan Goode-Null's (2002) study of childhood health and development in the NYABG sample found that the enslaved people

brought into New York between the years of 1664 to 1741 were largely from the Caribbean. Following McManus' *A History of Negro Slavery in New York* (1966), Goode-Null explains that from 1741 to 1770, due to the cessation of slave trading between the British and Spanish colonies and the fear that a slave revolt aborted in 1741 might repeat the events of the 1712 slave revolt in New York, enslaved Africans were imported directly from Africa, rather than via the Caribbean, and were largely young women ages 13 to 40 years and children preferably of nine to ten years of age, rather than adult males. Adult enslaved men from the Caribbean were considered the strategists behind the successful and aborted revolts (Goode-Null 2002: 28; see also chapter thirteen in this volume and the History Final Report for further reference to these factors).

These historical data suggest at least two additional interpretations. One explanation assumes that many children experiencing stress episodes during the ages of 3.5 to 6.5 years, and who lived to adulthood, were born within the colony of New York. Goode-Null's study reports that enslaved children in New York were frequently sold by the age of six years (History Final Report, 2004; Goode-Null 2002: 37-38). Advertisements indicate domestic skills promoting the marketability of enslaved children. Therefore, it is likely that children approaching the age of six years may have experienced trauma related to separation from their parents, differential nutrition provisions provided by non-parental custodians or slaveholders, or stresses and increased exposure to disease due to induction into domestic or other labor duties. Children under the age of 15 were highly stressed, and approaching the age of six may have been a significant stage within the life histories of children born within the legal status of "slave" in colonial New York. Furthermore, legal definitions of "adult" were applied to children

over the age of ten years in the 1731 and 1737/8 censuses, and at sixteen years in the census data prior to 1731 and after 1737/8, including the 1810 census (Chapter 13; Goode-Null 2002; Blakey 1998: 62). This legal status as "adult" would most likely have effected the character of labor expected of young enslaved Africans under the age of 15 and within the age group of 15 to 24. These data further suggest that a child approaching the age of nine or ten may be prepared for an occupational position through entry into labor training and work. Substantial third molar defect frequencies, especially for those who died between 15 and 24 years of age, characterize stresses of older children and adolescents whether or not they were born in New York.

A second interpretation assumes the inclusion of children imported from Africa to New York, again around the age of nine or ten, as enslaved laborers. These children may have experienced high levels of physiological stress during their earlier childhood related to shifts in political power and socioeconomic upheaval within the Atlantic slave trade networks that may have factored into their enslavement. Also, children under the age of 15 years could likely have experienced the middle passage prior to their arrival in New York. Any of a host of other possible inadequacies of the large, stratified agrarian societies from which they derived may have contributed to moderately high hypoplastic frequencies in the childhoods of those who died as adults in New York. Consistent with other findings of this study, most of the stresses shown by adult teeth were likely produced by factors within their native African environments with a minority of the adult teeth developing during childhoods in New York. The high third molar frequencies for those who died between 15 and 24 years of age also suggest effects deriving from arrivals in New York between 9 and 16 years of age in at least 44 percent of the individuals.

Those who lived to old age showed far less stress during 9-16 years of age than those who died shortly after arrival in New York.

Our observation that those who lived the longest also had the lowest evidence of childhood stressors may suggest that higher chances of survival to adulthood are associated with having lower stress in childhood, irrespective of where the childhood took place. An attrition of hypoplastic individuals that is associated with age has been postulated elsewhere (Blakey and Armelagos 1985). These are not mutually exclusive propositions; those born in Africa may have had fewer childhood stressors and survived to older ages at death in New York than those who were born in New York City.

One approach to this question has been to compare hypoplasia frequencies for individuals with culturally-modified teeth to those without such modifications (see Figure 8.7 and Table 8.10). Handler's historical study (1994) and our chemical research (see Chapter 6) strongly suggest that modified teeth most frequently indicate African birth. Individuals without cultural modification (probably both African and non-African born) had higher frequencies of hypoplasia than individuals without [Modified 66.7% (n=6); Unmodified 72.9% (n=40)].

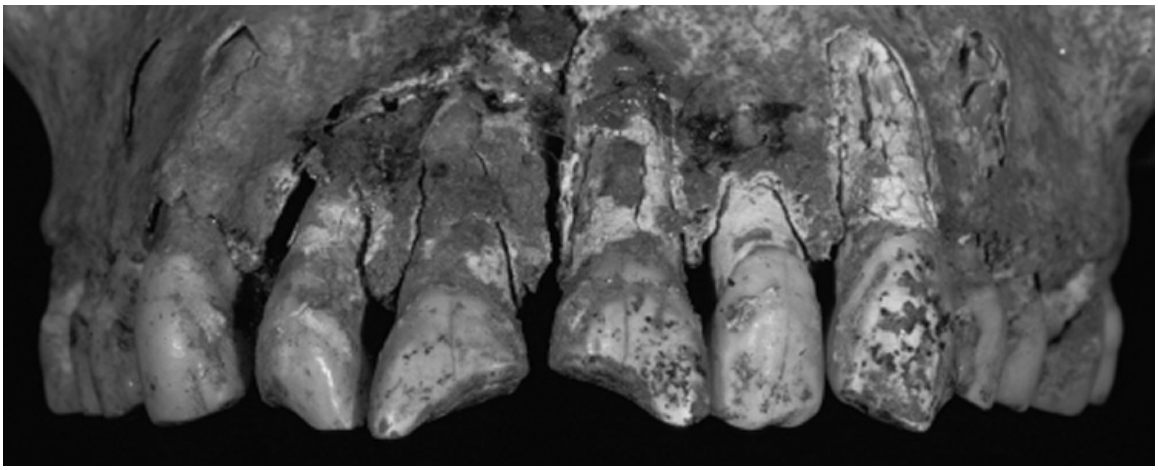

Figure 8.7: Dental Modification

The mean ages at death for individuals with modified and unmodified teeth were comparable, though slightly older for individuals with modified teeth (34 years for individuals with modified teeth and 31 years for individuals with unmodified teeth). While consistent with the association between African birth and lower defect frequencies, these differences are not statistically significant at the $p<.05$ level. Chemical and mtDNA analyses will provide greater insight into these interpretations. Indeed, chemical sourcing data would add greatly to the conclusiveness of these tests by providing an independent method of identifying place of birth in at least two hundred ABG individuals; this should be done in a future study.

Table 8.10: NYABG Hypoplasia in Culturally Modified and Unmodified Permanent Teeth

Culturally Modified (n=9)	Unmodified (n=56)
66.7% (n=6)	71.4% (n=40)

The highest levels of hypoplasia are found within the individuals with deciduous dentition and may therefore represent effects of prenatal stress experienced by the mother during pregnancy. Furthermore, the decreasing frequencies of hypoplasia exhibited by individuals who lived longer suggest a relationship between stress episodes indicated by hypoplasia and a decreased life span.

Dental Enamel Hypocalcification

A study of dental enamel hypocalcification was conducted to assess frequencies within a subsample of 99 individuals. This subject had permanent dentition, including a left or right maxillary central incisor and a left or right mandibular canine, and included children with deciduous left or right maxillary incisors, left or right mandibular canines, and a second molar.

Within this study of the ABG sample, 67.6 percent (n=29) of the 34 children with deciduous dentition had hypocalcification. Among the 65 individuals with permanent dentition, 18.5 percent (n=12) had hypocalcification (see Table 8.11). Women had a higher frequency of hypocalcification than did men (72.7% of the 24 females versus 27.3% of the 35 males).

Within this subsample, 60.5 percent (n=23) of the 38 children under the age of 15 years had hypocalcification, whereas only 10 percent (n=2) of the 20 young adults aged 15 to 24.9 years and 28.6 percent (n=10) of the adults aged 25 and older had hypocalcification (see Table 8.11). This difference was statistically significant (Pearson chi-square = 19.84, 2 df, $p < .0005$) and mainly reflects the change from predominantly primary to secondary teeth by age 15. The difference between hypocalcification frequencies found in individuals with deciduous dentition (67.6% n=23) and permanent dentition (18.5% n=12) should not be considered in the same manner in which this age-related pattern in hypoplasia has been considered.

Deciduous dentition is more likely to become hypocalcified than to exhibit hypoplasia, and deciduous dentition typically displays higher frequencies of hypocalcification in comparison to permanent dentition (Blakey et al. 1997). Thus, the observed low frequency of hypocalcification in permanent dentition follows the expected pattern due to suspected intrinsic differences between deciduous and permanent dentition that may have nothing to do with stressor prevalence. Comparisons of hypocalcification across primary and secondary dentition are therefore inappropriate.

Table 8.11: NYABG Comparison of Hypocalcification and Hypoplasia Frequencies by Age Group (N=99)

Age Group	Within Age Group	
	Hypocalcification	**Hypoplasia**
1.0- 14.9	60.5% (n=23)	86.5% (n=32)
15.0-24.9	10% (n=2)	80.0% (n=16)
25.0-55 +	28.6% (n=10)	66.7% (n=30)

Comparison of the two defect types within deciduous dentitions is of interest. Deciduous dentition forms in-utero and continues into the first year of life and therefore represents early childhood development and a measure of prenatal health and the health status of the mother. Hypocalcification and hypoplasia frequencies were both highest in children dying prior to the age of 15 years, demonstrating high physiological stress and vulnerability during the prenatal and early childhood years. The higher levels of hypoplasia (86.5%) versus hypocalcification (65.7%) within deciduous dentition (n=34) is unexpected, however, given the tendency of deciduous teeth to preferentially exhibit hypocalcification. Hypoplasia frequency in this case is extraordinarily high compared to other deciduous dental studies using similar methods (Blakey and Armelagos 1985; Blakey et al. 1995; 1997; Rankin-Hill 1997). Both defect frequencies indicate the extremely high levels of stress experienced in-utero and during the first year of life among the ABG children who died before the age of fifteen.

Conclusions

Historical data on the ages of children who were in various stressful contexts have been applied to explain developmental defect frequencies that occurred at different ages in the childhood and adolescent periods of the life cycle. Children, likely born in colonial New York within the condition of slavery, were more vulnerable to health risks and early

death due to nutritional deficiencies and illness than is evident for the childhoods of those who were likely to have been born in Africa. The findings of this study suggest disparity between early childhood health and nutrition for individuals more likely to have been born in colonial New York and individuals likely to have been born as free people in the agricultural villages of the war-torn states of West and Central Africa (see *History Final Report*, section I). The fact that higher frequencies of enamel defects were shown to exist among children under the age of fifteen and among individuals without dental modification, than among individuals who were most likely to have been born in Africa (older individuals and those with modified teeth), supports this hypothesis. The chronology of physiological insults resulting in hypoplasia further supports the vulnerability of childhood and adolescence for enslaved Africans in New York.

The third molar data reflect the trajectory of life experience for individuals, most of whom were likely to have been born in Africa and enslaved in the Americas. Significantly higher hypoplasia frequencies found in the third molars representing the developmental period of 9 through 16 years correspond with historical data indicating high levels of importation of older children, adolescents and young adults in the eighteenth-century. These findings indicate that the quality of life for Africans was greatly compromised upon entry into the New York environment of enslavement through the processes of either birth or forced migration.